PERGAMON

European Journal of Cancer 38 (2002) 1769–1774

European Journal of Cancer

www.ejconline.com

Body size indices at different ages and epithelial ovarian cancer risk

L. Dal Maso[a,*], S. Franceschi[b], E. Negri[c], E. Conti[d], M. Montella[e], S. Vaccarella[a], V. Canzonieri[f], F. Parazzini[c], C. La Vecchia[c,g]

[a]*Servizio di Epidemiologia, Centro di Riferimento Oncologico, Aviano (PN), Italy*
[b]*Field and Intervention Studies Unit, International Agency for Research on Cancer, Lyon, France*
[c]*Istituto di Ricerche Farmacologiche "Mario Negri", Milan, Italy*
[d]*Servizio di Epidemiologia e Oncogenesi, Istituto Regina Elena per lo Studio e la Cura dei Tumori, Rome, Italy*
[e]*Servizio di Epidemiologia, Istituto Tumori "Fondazione Pascale", Napoli, Italy*
[f]*Divisione di Anatomia Patologica, Centro di Riferimento Oncologico, Aviano (PN), Italy*
[g]*Istituto di Statistica Medica e Biometria, Università degli Studi di Milano, Milan, Italy*

Received 21 November 2001; received in revised form 18 March 2002; accepted 26 March 2002

Abstract

The relationship between body mass measures at diagnosis and/or at different ages and ovarian cancer risk was investigated using an Italian multicentre case–control study. The study, conducted between 1992 and 1999, included 1031 cases of incident, histologically-confirmed epithelial ovarian cancer and 2411 controls admitted to the same network of hospitals for acute non-neoplastic conditions. Odds ratios (OR) and 95% confidence intervals (CI) were obtained using unconditional multiple logistic regression analyses. Weight and body mass index (BMI, kg/m^2) 1 year prior to diagnosis/interview were not associated with ovarian cancer risk. A direct association emerged with waist-to-hip ratio (W/H) (OR = 1.45 in the highest category), particularly among women with stage I-II cancers. Cases also had a higher BMI at age 30 years (OR = 1.22). Conversely, cases had lower weight gain between age 30 years and the year prior to diagnosis/interview, both for cases with stage I-II and those with stage III-IV cancers.

Keywords: Case–control study; Ovarian cancer; Body mass index

1. Introduction

Overweight and obesity account for 39% of endometrial carcinomas and 9% of postmenopausal breast carcinomas in Europe [1]. Conversely, the relationship between weight and ovarian cancer risk is not clear, chiefly on account of the difficulty of allowing for disease-related weight loss in retrospective (case–control) studies. Some case–control studies [2–5] showed that women with ovarian cancer were leaner than controls. In others, however, a moderate positive association with usual adult weight or body mass index (BMI) was reported [6,7]. In three studies [8–10], some excess risk was largely confined to the heaviest women. In a study where BMI years before diagnosis was evaluated [10], the direct association with excess weight was restricted to women who were premenopausal and who had serous or endometrioid ovarian carcinomas. Most case–control [11–15] and two cohort studies [16,17], however, did not show a consistent association between weight or BMI and ovarian cancer risk. A recent paper [18] summarised results on the association between body size and ovarian cancer by study design. Different patterns emerged in case–control studies and population-based studies which showed a modest direct association. However, an interpretation of the results is hampered by substantial differences in the definition of those who were overweight in hospital-based case–control studies that showed association in both directions.

Some [2,8,19,20], but not all case–control studies [11], found that women with ovarian cancer were slightly taller than control women. Finally, a moderate association emerged from a few studies [17,21] in women with an elevated waist-to-hip ratio (W/H) (i.e. android fat distribution).

* Corresponding author. Tel.: +39-0434-659354; fax: +39-0434-659222.
E-mail address: epidemiology@cro.it (L. Dal Maso).

0959-8049/02/$ - see front matter
PII: S0959-8049(02)00155-7

To provide further information on the issue, we analysed data from a uniquely large case–control study of epithelial ovarian cancer conducted in four Italian areas between 1992 and 1999. Our study included extensive information on several anthropometric factors and weight at various ages, as well as on major potential confounding factors.

2. Patients and methods

The data were derived from a case–control study of ovarian cancer, conducted between January 1992 and September 1999 in four Italian areas: greater Milan, the provinces of Pordenone, Padua and Gorizia (north-eastern Italy); the province of Latina (central Italy); the urban area of Naples (southern Italy) [22]. The interviewers were centrally trained. Less than 4% of cases and controls who were approached refused to be interviewed, and the response rates did not vary across hospitals and geographical areas.

Cases were 1031 women (median age 56, range 18–79 years) with incident (i.e. diagnosed within the year before interview), histologically-confirmed, epithelial ovarian cancer, admitted to the major teaching and general hospitals in the areas under surveillance. Information on stage according to the International Federation of Gynecology and Obstetrics (FIGO) classification [23] was available for 587 (57%) ovarian cancer cases, chiefly from the provinces of Pordenone and Padua. The majority of cases (78%) had an advanced stage (III or IV) disease.

Controls were 2411 women (median age 57, range 17–79 years) who lived in the same geographical areas as the cancer cases and were admitted to hospitals that shared the same catchment areas as those where cases were referred to. A wide spectrum of acute conditions unrelated to known or potential risk factors for ovarian cancer was the reason for admission. Women were not eligible as controls if they were admitted for hormonal and gynaecological diseases, and if they had undergone a bilateral oophorectomy. Among the controls, 26% had traumatic conditions (mostly fractures and sprains), 28% non-traumatic orthopaedic disorders (mostly lower back pain and disc disorders), 15% acute surgical conditions (mostly abdominal, such as acute appendicitis or strangulated hernia), and 31% other miscellaneous illnesses such as eye, ear, nose, throat and dental disorders.

All interviews were conducted in a hospital setting using a structured questionnaire which included information on age, education and other socio-economic factors, physical activity, smoking habit, alcohol intake, coffee consumption, a detailed food frequency section, a problem-oriented medical history, history of cancer in first degree relatives, gynaecological and obstetrical data, and history of the use of oral contraceptives (OCs) and hormone replacement therapy. Menstrual and reproductive histories were also obtained.

BMIs measures at different ages were examined in a detailed section of the questionnaire. Study subjects were asked to report their height and habitual weight 1 year prior to the cancer diagnosis or interview (in controls). In addition, weight at 30 and 50 years of age, highest and lowest weight in adult life (pregnancy period excluded) and bra cup size in young adulthood were elicited. Perceived body size at 12 years of age was also explored (i.e. thinner than, same as, heavier than peers). The interviewers measured the circumference of the waist (2 cm above the umbilicus) and hips (maximal protrusion). In 30% of ovarian cancer cases and 23% of control subjects, waist and hip measurements could not be obtained for various logistical reasons.

2.1. Statistical analysis

BMI was calculated as weight/height^2 (kg/m^2) and W/H as the ratio between the circumferences of the waist and hip. Odds ratios (OR) of ovarian cancer, and the corresponding 95% confidence intervals (CI), for various levels of body mass measures were derived using unconditional multiple logistic regression, fitted by the method of maximum likelihood [24]. The effect of several potential confounding factors was considered, i.e. beside design factors (study centre and age in 5-year groups), education, parity and OC use. Additional adjustment for total calorie intake, menopausal status and family history of ovarian/breast cancer in first-degree relatives did not modify our findings. Tests for linear trend were assessed by means of the Wald Chi-square on the variables considered as categorical.

3. Results

Table 1 shows the distribution of ovarian cancer cases and controls, with the corresponding ORs according to various body size measurements 1 year prior to the diagnosis/interview. An inverse association with ovarian cancer risk emerged for height (OR = 0.72 in highest versus lowest quintile; 95% CI: 0.56–0.93). Weight and BMI did not show clear patterns of risk. ORs were 0.99, 0.76, and 1.07 for BMIs of 21 to <25, 25 to <30 and 30 or higher, respectively, compared with a BMI of less than 21 (Table 1). Bra cup size was unrelated to the ovarian cancer risk, whereas W/H showed a non-linear association with lowered ORs in the intermediate quintiles and an elevated one (OR = 1.45, 95% CI: 1.07–1.96) at the $\geqslant 0.89$ level (Table 1).

Body size measurements at different ages are shown in Table 2. No association with ovarian cancer risk emerged for perceived body size at 12–15 years compared with one's peers. In addition BMI at age 50 years

Table 1
Distribution of 1031 cases of ovarian cancer and 2411 controls, odds ratios (OR)[a] and corresponding 95% confidence intervals (95% CI), according to various body-size measurements. Italy, 1992–1999[b]

	Cases	Controls	OR	(95% CI)
Height (cm)				
⩽155	239	480	1	
156–159	170	317	1.07	(0.82–1.39)
160–161	183	510	0.72	(0.56–0.92)
162–165	246	598	0.84	(0.66–1.06)
⩾166	188	494	0.72	(0.56–0.93)
χ^2trend			8.49	$P<0.01$
Weight (kg)				
⩽54	186	390	1	
55–60	229	531	0.93	(0.72–1.19)
61–66	177	500	0.66	(0.50–0.86)
67–74	183	478	0.70	(0.53–0.92)
⩾75	250	506	0.88	(0.68–1.14)
χ^2trend			2.13	$P=0.14$
Body mass index (kg/m^2)				
<21	143	346	1	
21 to <25	406	920	0.99	(0.77–1.27)
25 to <30	299	810	0.76	(0.58–0.99)
⩾30	173	318	1.07	(0.79–1.44)
χ^2trend			0.39	$P=0.53$
Bra cup size				
⩽II	259	588	1	
III	362	896	0.88	(0.72–1.09)
IV	222	521	0.86	(0.68–1.08)
⩾V	180	371	0.89	(0.69–1.15)
χ^2trend			0.96	$P=0.33$
Waist-to-hip ratio[c]				
⩽0.76	155	365	1	
0.77–0.80	103	367	0.59	(0.43–0.81)
0.81–0.84	133	383	0.76	(0.56–1.03)
0.85–0.88	126	381	0.69	(0.50–0.94)
⩾0.89	209	351	1.45	(1.07–1.96)
χ^2trend			9.11	$P<0.01$

[a] Estimates from multiple logistic regression equations, including terms for age (quinquennia), study centre, education, parity and oral contraceptive use.
[b] The sum may not add up to the total because of some missing values.
[c] As in model [a] including terms for body mass index.

and maximal BMI in a life time seemed to be uninfluential. Conversely, ovarian cancer cases showed a slightly higher BMI than control women at age 30 years (OR = 1.16 for 21 to <25 versus <21 kg/m^2; 95% CI: 0.96–1.40 and OR = 1.22; 95% CI: 0.96–1.55 for those ⩾25 kg/m^2) and a more elevated minimal BMI lifetime (OR for minimal BMI 20 to <22 versus <20 kg/m^2 = 1.19; 95% CI: 0.98–1.44 and OR = 1.27; 95% CI:

Table 2
Distribution of 1031 cases of ovarian cancer and 2411 controls, odds ratios (OR) [a] and corresponding 95% confidence intervals (95% CI), according to body-size measurements at different ages. Italy, 1992–1999[b]

	Cases	Controls	OR	(95% CI)
Perceived body-size at 12–15 years				
Thinner	361	787	1	
Same	408	1030	0.94	(0.78–1.12)
Heavier	262	594	0.93	(0.76–1.14)
χ^2trend			0.51	$P=0.48$
BMI at age 30[c] (kg/m^2)				
<21	276	801	1	
21 to <25	443	973	1.16	(0.96–1.40)
⩾25	207	410	1.22	(0.96–1.55)
χ^2trend			3.00	$P=0.08$
BMI at age 50[d] (kg/m^2)				
<21	62	183	1	
21 to <25	228	593	0.99	(0.70–1.41)
25 to <30	181	406	0.96	(0.66–1.39)
⩾30	61	119	1.02	(0.64–1.62)
χ^2trend			0.00	$P=0.96$
Maximal BMI (kg/m^2)				
<25	400	903	1	
25 to <30	356	945	0.78	(0.65–0.94)
⩾30	254	514	0.95	(0.77–1.18)
χ^2trend			0.55	$P=0.46$
Minimal BMI (kg/m^2)				
<20	392	1124	1	
20 to <22	301	637	1.19	(0.98–1.44)
⩾22	302	571	1.27	(1.04–1.55)
χ^2trend			6.01	$P=0.01$
Increase of BMI from age 30[c] (kg/m^2)				
⩽0	228	467	1	
>0 to <4	465	1001	0.94	(0.76–1.15)
⩾4	233	716	0.62	(0.49–0.78)
χ^2trend			17.5	$P<0.01$
Increase of BMI from age 30 to 50[d] (kg/m^2)				
<1	198	411	1	
1 to <3	173	447	0.81	(0.62–1.06)
⩾3	145	404	0.68	(0.52–0.90)
χ^2trend			7.49	$P<0.01$

BMI, body mass index.
[a] Estimates from multiple logistic regression equations, including terms for age (quinquennia), study centre, education, parity and oral contraceptive use.
[b] The sum may not add up to the total because of some missing values.
[c] For subjects aged 35 years or more; ($n=926$ cases, 2184 controls).
[d] For subjects aged 55 years or more; ($n=532$ cases, 1301 controls).

1.04–1.55 for those $\geqslant 22$ kg/m^2. Thus, ovarian cancer cases seemed to have experienced, on average, a lower weight gain from age 30 years (OR = 0.62 for 5 versus <1 BMI unit gain; 95% CI: 0.49–0.78) or from age 30 to 50 years (OR = 0.68; 95% CI: 0.52–0.90).

Since advanced ovarian cancer is often accompanied by weight loss and ascites, the major aforementioned findings were re-assessed separately among the stage I-II patients, who are generally unaffected by these factors, and the III-IV ones (Table 3). A BMI equal to or greater than 30 was associated with an OR of 1.56 (95% CI: 0.75–3.23) among stage I-II patients, but of 1.09 (95% CI: 0.74–1.60) among the stage III-IV ovarian cancer cases. The U-shaped relationship between W/H and ovarian cancer risk showed in Table 1 seemed thus to be chiefly attributable to cases with advanced disease. Among stage I-II ovarian cancer cases, the increase of risk with the increase in W/H was nearly linear (OR = 1.93 for W/H $\geqslant 0.89$ versus $\leqslant 0.76$; 95% CI: 0.93–4.01). The inverse association of risk with weight gain between age 30 years and the year prior to diagnosis/interview was confirmed among both stage I-II (OR = 0.68) and stage III-IV ovarian cancer cases (OR = 0.66).

The apparent effect of various body size measurements was consistent across different strata of age, study centre, and in pre- and postmenopausal women (data not shown).

4. Discussion

Our study provides further evidence that weight is not strongly associated with the risk of epithelial ovarian cancer. Most studies, in particular population-based case–control studies [11–18] showed a modest direct association with weight, but it remains difficult to allow for the influence of weight loss caused by ovarian cancer. Findings are, thus, compatible with a modest association between excess weight and epithelial ovarian cancer of the magnitude found for postmenopausal breast cancer [25]. The major contribution of our study is the new evidence added to the lack of association with weight at different periods of life, including one year prior to cancer diagnosis/interview, adolescence and at age 50 years.

From a biological viewpoint, a difference between epithelial ovarian cancer and endometrial or postmenopausal breast cancer is not surprising. Excessive

Table 3
Distribution of ovarian cancer cases and controls, odds ratios (OR)[a] and corresponding 95% confidence intervals (95% CI), according cancer stage and waist-to-hip ratio, Italy, 1992–1999[b]

	Stage I-II			Stage III-IV			Controls
	Cases	OR	(95% CI)	Cases	OR	(95% CI)	
Body mass index							
<21	16	1		68	1		346
21 to <25	44	0.99	(0.52–1.86)	166	0.85	(0.62–1.18)	920
25 to <30	40	1.03	(0.53–2.00)	142	0.78	(0.55–1.10)	810
$\geqslant 30$	24	1.56	(0.75–3.23)	82	1.09	(0.74–1.60)	318
	χ^2trend	1.66	$P = 0.20$		0.09	$P = 0.77$	
Waist-to-hip ratio[c]							
$\leqslant 0.76$	15	1		63	1		365
0.77—0.80	12	0.66	(0.29–1.49)	39	0.51	(0.33–0.80)	367
0.81—0.84	22	1.12	(0.53–2.34)	52	0.62	(0.41–0.95)	383
0.85—0.88	26	1.36	(0.66–2.83)	58	0.67	(0.44–1.03)	381
$\geqslant 0.89$	29	1.93	(0.93–4.01)	100	1.32	(0.89–1.97)	351
	χ^2trend	6.48	$P = 0.01$		5.19	$P = 0.02$	
Increase of BMI from age 30[d] (kg/m^2)							
$\leqslant 0$	27	1		99	1		467
0 to <4	48	0.95	(0.57–1.59)	212	1.06	(0.80–1.40)	1001
$\geqslant 4$	28	0.68	(0.39–1.22)	109	0.66	(0.49–0.90)	716
	χ^2trend	1.78	$P = 0.18$		7.92	$P < 0.01$	

[a] Estimates from multiple logistic regression equations, including terms for age (quinquennia), study centre, education, parity and oral contraceptive use.
[b] The sum may not add up to the total because of some missing values.
[c] As in model [a] including terms for body mass index.
[d] For subjects aged 35 years or more.

weight entails, in postmenopausal women, increased levels of circulating oestrogens [28]. The ovary, however, is a source of oestrogens, but not an obvious target of them. Receptors to progesterone and androgens seem to be more often present in epithelial ovarian cells than oestrogen receptors [26]. Furthermore, the ovarian surface epithelium is avascular, suggesting a largely paracrine rather than endocrine influence of hormonal factors. Within the ovaries, the secretion of androgens is higher than that of oestrogens [26]. Finally, at variance with the endometrium and the breast, where oestrogens can prolong the activity of the tissue beyond the cessation of ovarian activity [26], no level of endogenous or exogenous oestrogens can prolong ovulations (and, thus, cancer risk) after menopause [6].

Within these overall negative findings, however, a few interesting risk patterns emerged and are worth discussing. Women with an android pattern of fat deposition were at a moderately increased risk. W/H is especially hard to evaluate in ovarian cancer patients as weight loss selectively lowers, but ascites increases, the W/H. Our findings could, however, be confirmed in a small subset of cases with stage I-II ovarian cancers, where weight loss or ascites are rare. They are also in agreement with the results of a prospective investigation [17], where a high W/H (greater than 0.89 versus lower than 0.78) was associated with a OR of 1.9. A high W/H is associated with hyperinsulinaemia, hyperandrogenaemia, and polycystic ovaries [26]. It is possible that one or more of these three factors may increase ovarian cancer risk. In our study, a diet high in refined carbohydrates, which can lead to hyperinsulinaemia and insulin resistance, was also related to an increased risk of ovarian cancer [22].

As we were able to evaluate the lifetime history of weight, we have also found that ovarian cancer cases may differ subtly from control women in having experienced less weight gain and/or fewer weight fluctuations in their adult life. Their lowest BMI ever was higher than the one among control women and their BMI increase between young adulthood and diagnosis/interview more modest. If confirmed, these findings may point to the role of a relatively constant weight across adult life in maintaining 'incessant ovulations' and, thus, enhancing the probability of ovarian tumours [6].

Finally, height was inversely associated with ovarian cancer risk in our study. Such findings were not accounted for by a different geographical origin of our hospital-based cases and controls, since we carefully adjusted for the place of living of the study subjects. It is unclear whether height may be in Italy a proxy of dietary intake in adolescents (i.e. in the 1940s and 1950s, for the majority of study women) or whether the present unexpected association with height should be considered due to chance.

Problems of reliability in the anthropometric measures could not be excluded [27]. Weights at different ages, in our study, had to be self-reported ones. However, they are generally well correlated with corresponding measures even in older persons [28] and there is no evidence that self-reported measures are biased between cases and controls [29]. Moreover, the similar interview settings provide some reassurance against a potential differential recall [30].

This study was not population-based, but cases were identified in the major teaching and general hospitals of the areas under surveillance, and the participation of cases and controls was almost complete. We excluded from the control group patients admitted to hospital for chronic diseases and for any gynaecological conditions, and exclusion from the analysis of orthopaedic (including traumatic) control women, who may have been more often overweight than the general population, did not materially modify the risk estimates.

The major strength and originality of this study is related to the uniquely large number of ovarian cancer cases interviewed and to the data available on a wide range of anthropometric measures at various ages and on cancer stage and major potential confounding factors.

In conclusion, our study suggests that even though there appears to be a lack of an important role for recent weight, an elevated W/H and a tendency to maintain a constant weight during adulthood and middle-age are directly correlated with the risk of epithelial ovarian cancers.

Acknowledgements

This work was conducted with the contribution of the Italian Association and the Italian League for Cancer Research, Milan. The Authors thank Mrs Ilaria Calderan for her editorial assistance.

References

1. Bergstrom A, Pisani P, Tenet V, *et al*. Overweight as an avoidable cause of cancer in Europe. *Int J Cancer* 2001, **91**, 421–430.
2. Mori M, Kiyosawa H, Miyake H. Case-control study of ovarian cancer in Japan. *Cancer* 1984, **53**, 2746–2752.
3. Mori M, Harabuchi I, Miyake H, *et al*. Reproductive, genetic, and dietary risk factors for ovarian cancer. *Am J Epidemiol* 1988, **128**, 771–777.
4. Engle A, Muscat JE, Harris RE. Nutritional risk factors and ovarian cancer. *Nutr Cancer* 1991, **15**, 239–247.
5. Parazzini F, Moroni S, La Vecchia C, *et al*. Ovarian cancer risk and history of selected medical conditions linked with female hormones. *Eur J Cancer* 1997, **33**, 1634–1637.
6. Casagrande JT, Louie EW, Pike MC, *et al*. "Incessant ovulation" and ovarian cancer. *Lancet* 1979, **2**, 170–173.
7. Purdie D, Green A, Bain C, *et al*. Reproductive and other factors and risk of epithelial ovarian cancer: an Australian case-control study. *Int J Cancer* 1995, **62**, 678–684.
8. Cramer DW, Welch WR, Hutchison GB, *et al*. Dietary animal fat

in relation to ovarian cancer risk. *Obstet Gynecol* 1984, **63**, 833–838.
9. Garfinkel L. Overweight and cancer. *Ann Intern Med* 1985, **103**, 1034–1036.
10. Farrow DC, Weiss NS, Lyon JL, Daling JR. Association of obesity and ovarian cancer in a case-control study. *Am J Epidemiol* 1989, **129**, 1300–1304.
11. Polychronopoulou A, Tzonou A, Hsieh C-C, *et al*. Reproductive variables, tobacco, ethanol, coffee and somatometry as risk factors for ovarian cancer. *Int J Cancer* 1993, **55**, 402–407.
12. Beard CM, Hartmann LC, Atkinson EJ, *et al*. The epidemiology of ovarian cancer: a population-based study in Olmsted Country, Minnesota, 1935–1991. *Ann Epidemiol* 1935, **2000**, 10 14–23.
13. Greggi S, Parazzini F, Paratore MP, *et al*. Risk factors for ovarian cancer in central Italy. *Gynecol Oncol* 2000, **79**, 50–54.
14. Ness RB, Grisso JA, Klapper J, *et al*. Risk of ovarian cancer in relation to estrogen and progestin dose and use characteristics of oral contraceptives. *Am J Epidemiol* 2000, **152**, 233–241.
15. Siskind V, Green A, Bain C, Purdie D. Beyond ovulation: oral contraceptives and epithelial ovarian cancer. *Epidemiology* 2000, **11**, 106–110.
16. Törnberg SA, Carstensen JM. Relationship between Quetelet's index and cancer of breast and female genital tract in 47,000 women followed for 25 years. *Br J Cancer* 1994, **69**, 358–361.
17. Mink PJ, Folsom AR, Sellers TA, Kushi LH. Physical activity, waist-to-hip ratio, and other risk factors for ovarian cancer: a follow-up study of older women. *Epidemiology* 1996, **7**, 38–45.
18. Purdie DM, Bain CJ, Webb PM, *et al*. Body size and ovarian cancer: case-control study and systematic review (Australia). *Cancer Causes Control* 2001, **12**, 855–863.
19. Hildreth NG, Kelsey JL, Livolsi VA, *et al*. An epidemiologic study of epithelial/carcinoma of the ovary. *Am J Epidemiol* 1981, **114**, 398–405.
20. Tzonou A, Day NE, Trichopoulos D, *et al*. The epidemiology of ovarian cancer in Greece: case-control study. *Eur J Cancer* 1984, **20**, 1045–1052.
21. Lapidus L, Helgesson O, Merch C, Bjorntorp P. Adipose tissue distribution and female carcinomas. A 12-year follow-up of participants in the population study of women in Gothenburg, Sweden. *Int J Obes* 1988, **12**, 361–368.
22. Bosetti C, Negri E, Franceschi S, *et al*. Diet and ovarian cancer risk: a case-control study in Italy. *Int J Cancer* 2001, **93**, 911–915.
23. Pecorelli S, Benedet JL, Creasman WT, Shephered JH. FIGO staging of gynecologic cancer. 1994–1997 FIGO Committee on Gynecologic Oncology. International Federation of Gynecology and Obstetrics. *Int J Gynaecol Obstet* 1999, **64**, 5–10.
24. Breslow NE, Day NE. *Statistical Methods in Cancer Research, Vol. 1. The Analysis of Case-control Studies. IARC Sci. Publ., No. 32*. Lyon, IARC, 1980.
25. Collaborative Group on Hormonal Factors in Brest Cancer. Breast cancer and hormone replacement therapy: collaborative reanalysis of data from 51 epidemiological studies of 52,705 women with breast cancer and 108,411 women without breast cancer. *Lancet* 1997, **350**, 1047–1059.
26. Risch HA. Hormonal etiology of epithelial ovarian cancer, with a hypothesis concerning the role of androgens and progesterone. *J Natl Cancer Inst* 1998, **90**, 1774–1786.
27. Frost C, Coleman MP. Obesity and ovarian cancer. *Eur J Cancer* 1997, **33**, 1529–1531.
28. Klipstein-Grobusch K, Kroke K, Boeing H. Reproducibility of self-reported past body weight. *Eur J Clin Nutr* 1998, **52**, 525–528.
29. Casey VA, Dwyer JT, Berkey CS, *et al*. Long-term memory of body weight and past weight satisfaction: a longitudinal follow-up study. *Am J Clin Nutr* 1991, **53**, 1493–1498.
30. D'Avanzo B, La Vecchia C, Katsouyanni K, *et al*. An assessment, and reproducibility of food frequency data provided by hospital controls. *Eur J Cancer Prev* 1997, **6**, 288–293.